ADVERSE EFFECTS OF ADJUVANTS IN VACCINES

Adjuvants are chemical substances which are added to vaccines to boost immune response, but they can also cause a wide range of adverse side effects.

Part 2 of 2

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[Editor's Note: This article refers to research studies involving animals. We wish to advise readers that we at NEXUS do not condone or support the validity, efficacy or morality of animal experimentation or vivisection.]

IMMUNOLOGY PRINCIPLES: ANTIBODY RESPONSE

To explain the action of adjuvants, we should look into immunology. The theory of vaccine efficacy is based on the ability of vaccines to evoke the formation of antibodies. This is of varying efficacy, depending on the nature of the antigen(s) and the amount of antigenic substance administered.

However, the mechanisms for the diversity of immune reactions are complex, and to this day are not quite known and understood. There are numerous theories, the favoured one being antibody response as the sign of immunisation (acquiring immunity).

Specific immunity to a particular disease is generally considered to be the result of two kinds of activity: the humoral antibody and the cellular sensitivity.

The ability to form antibodies develops partly *in utero* and partly after birth in the neonatal period. In either case, immunological competence—the ability to respond immunologically to an antigenic stimulus—appears to originate with the thymic activity.

The thymus initially consists largely of primitive cellular elements which become peripheralised to the lymph nodes and spleen. These cells give rise to lymphoid cells, resulting in the development of immunological competence. The thymus may also exert a second activity in producing a hormone-like substance which is essential for the maturation of immunological competence in lymphoid cells. Such maturation also takes place by contact with thymus cells in the thymus.

Stimulation of the organism by antigen results in proliferation of cells of the lymphoid series accompanied by the formation of immunocytes, and this leads to the antibody production. Certain lymphocytes and possibly reticulum cells may be transformed into immunoblasts, which develop into immunologically active ("sensitised") lymphocytes and plasmocytes (plasma cells). Antibody formation is connected with plasma cells, while cellular immunity reactions are mainly lymphocytic.

None of the theories for antibody formation comprehends all the biological and chemical data now available. However, several principal theories have been considered at length.

The so-called *instructive theory* holds that the antigen is brought to the locus of antibody synthesis and there imposes in some way the synthesis of the specific antibody with reactive sites which are complementary to the antigen.

The *clonal selection theory*, evolved by Burnett (1960), presupposes that the information requisite to the synthesis of the antibody is part of the genetics. While the body develops a wide range of clones of cells necessary to cover all antigenic determinants by random mutation during early embryonic life, those clones which are capable of reacting with antigens of the body ("self") are destroyed, leaving only those cells which are not oriented to self ("non-self"). Upon stimulation by a foreign antigen, the clones of the cells corresponding to the particular foreign antigen are stimulated to proliferate and to produce the antibody.

Other researchers demonstrated that there are at least four different antigens formed by descendants of a single cloned cell. By this mechanism, the information for antibody synthesis is contained in the genetic material of each cell (DNA) but is normally repressed. The antigen then assumes the role of a de-repressor and initiates (provokes) the RNA synthesis for a particular messenger, resulting in the corresponding antibody production. The

antigen would instruct the genetically predisposed capability of multipotential cells as to which antibody to produce and might also command the cells to proliferate, resulting in clones of properly instructed cells.

There are two possible mechanisms for the elimination of antibodies against self: *immunological nonresponsiveness* and *immunological paralysis*. There are several states of immunological nonresponsiveness; one is illustrated by the exposure of a foetus or newborn to an antigen prior to the development of its ability to recognise the antigen as non-self (*immunological incompetence*). Immunological paralysis results from the injection of a very large amount of antigen into immunologically competent individuals. Nonspecific immunological suppression by cortisone, ACTH, nitrogen mustards and irradiation is also well known.

Cellular sensitivity, also known as *delayed* or *cellular hyper - sensitivity*, depends on the development of immunologically reactive or "sensitive" lymphocytes and possibly other cells which react with the corresponding antigen to give a typical delayed-type reaction after a period of several hours, days or even weeks.

Cellular hypersensitivity depends on the original antigenic stimulation and a *latent period*, and is specific in its response. Delayed-type hypersensitivity is characteristic of the body's response to various infectious agents such as viruses, bacteria, fungi, spirochetes and parasites. It is also characteristic of the body's response to various chemicals, such as mercury, endotoxins, antibiotics, various drugs and many other substances foreign to the body.

The inflammatory response which occurs in delayed-type hypersensitivity may not be protective, and in many instances may even be harmful.

The induction of a hypersensitivity reaction requires the presence in the tissues of the whole organism or certain derivatives of it, in addition to the specific antigen such as a lipid, in addition to tubercle bacillus protein. Sensitisation to a non-infectious substance must be mediated through the skin or mucuous membranes which probably provide further necessary co-factors.

A delayed hypersensitivity reaction may be enhanced experimentally by the employment of the antigen in a mineral oil adjuvant with added *Mycobacterium tuberculosis* or by injection of the antigen directly into the lymphatics. The delayed hypersensitivity response is accompanied by mild to severe inflammation which may cause cell injury and necrosis. *The inflammatory response which occurs in delayed-type hypersensitivity may not be protective, and in many instances may even be harmful* (e.g., rejection of grafts is directly linked to delayed hypersensitivity).

IMMUNOPATHOLOGY OF HYPERSENSITIVITY REACTIONS:

Immediate Hypersensitivity

This is the antibody-type reaction that is a secondary consequence to the beneficial effect of the combination of an antibody with its antigen.

Arthus-type Reaction

This reaction results from the precipitative union of a large amount of antigen with a highly reactive antibody in the blood vessels, and leads to vascular damage. The cascade of events includes spastic contraction of the arterioles, endothelial damage, formation of leukocyte thrombi, exudation of fluid and blood cells into the tissues, and sometimes ischemic necrosis. Periarteritis nodosa results from a similar antigen-antibody reaction and is characterised by inflammation of the smaller arteries and periarterial structures. It is accompanied by proliferation of the intima and two types of occlusion: (a) by proliferation or thrombosis; or (b) by the formation of nodules containing neutrophils and eosinophils.

Anaphylaxis

Injection of antigen and its combination with antibody may cause release from the cells (especially mast-cell fixed basophils) of physiologically active substances such as histamine, serotonin, acetylcholine, slow-reacting substances (SRS) and heparin. They act on smooth muscle and blood vessels and cause anaphylactic (hypersensitivity) shock, asthma attack, allergic oedema, rhinitis or hay fever, and accumulation of fluid in the joints.

Atopy

Atopy is caused by the union of antigen—usually pollens, dust, milk, wheat and animal danders—with a peculiar type of antibody (reagin). This reaction is relatively heat-labile and cannot be demonstrated by *in vitro* procedure. It has a special affinity for the skin and for familial predisposition to the disease. The reaction is nevertheless similar to other immediate-type sensitivities, with the release of histamine and its manifestation principally as asthma (breathing paralysis), hay fever, urticaria, angioedema and infantile eczema.

Delayed Hypersensitivity

The typical pathology of delayed hypersensitivity due to infectious agents involves perivascular infiltration of lymphocytes and histiocytes with the destruction of the antigen-containing parenchyma in the infiltrated area. The visual manifestations may vary from slight erythema and oedema to a violent reaction with progressive tissue destruction and necrosis. Local reactions include papular rose spots of typhoid fever, meningitis and a variety of infectious diseases, and contact sensitivities to plant and chemical substances manifesting as erythema, followed by papule and vesicle formation with resultant tissue damage and desquamation. Systemic reactions may accompany severe local reactions or may result from inhalation of the allergenic substances.

Humoral antibodies do not seem to play a role in delayed hypersensitivity reaction. The reactivity is transferred only by cells, presumably sensitised lymphocytes, and it is unlikely that histamine or other physiologically active substances play a role in the reaction. The reaction extends to any or all tissues where the offending antigen may occur.

Isoimmunological Disease

This is the result of an immunological reaction of a member of the same species to the tissue of another member of the same species. A blood transfusion reaction in a person given an incompatible blood type is a typical example. Another example is erythroblastosis fetalis, which results from the transfer of antibodies against the red blood cells of the foetus to the foetal circulation. Homograft rejection of tissues or organs between nonisologous members of a species is also immunologically based.

Immunological Disease Resulting from Adsorption of Foreign Substances

Under certain circumstances, foreign substances such as medications may combine with cells to render them antigenic. Subsequent exposure to such a foreign substance results in lytic, agglutinative or other types of cell-destructive activity. Such a reaction may involve red blood cells (drug-induced anaemias), platelets (drug-induced thrombocytopenic purpura), and leukocytosis (drug-induced agranulocytosis).

Bacteria or viruses may also alter cell surfaces by coating or by unmasking antigens through enzymatic activity which may render them vulnerable to immunological destruction.

Autoimmune Disease

Under certain circumstances, the body may respond immunologically to its own components or to intrinsic substances which are related antigenically to the host's own tissues. The circulating antibody or sensitised cells which are produced are then active in causing cellular injury to the tissues or organs of the body which bear the corresponding antigen.

Waksman (1962) proposed several mechanisms of autoimmunisation, such as:

1. Vaccination with organ-specific antigens which are isolated from the lymphatic channels and bloodstream and are not recognised as self when brought into contact with the immunologic process. They are represented in the central and peripheral nervous systems, lens, uvea, testes, thyroid (thyroglobulin), kidneys and other organs.

2. Vaccination against constituents of tissues which have been altered antigenetically by various factors. These include myocardial infarction, X-irradiation, enzymatic or other chemical alteration, and changes induced by infectious disease agents or by drugs. Erythrocytes, platelets and leucocytes are the most affected cells. Various organs may also be affected.

3. Vaccination with heterologous antigens which are sufficiently different to permit an immunological response but sufficiently alike to react with autologous antigens.

4. Alteration of the immunological apparatus so as to result in the failure of recognition of self. This occurs in neoplasia of the lymphatic system and in experimental grafting of immunologically competent heterologous lymphatic tissues under conditions which suppress the host's response to the graft and give rise to the wasting "runt disease" or "homologous disease".

5. Possible hereditary or other immunological abnormality. This is represented by a hyper-reactivity to antigens or other aberrations without apparent antigenic stimulation. Such mechanisms might be related to certain forms of the "collagen diseases", such as systemic lupus erythematosus in which there is an antibody against a diversity of antigens.

6. Experimentally, Freund's mineral oil adjuvant (usually with added mycobacteria) and certain bacteria or bacterial toxins may so alter the host as to bring about a ready response to unaltered normal homologous tissue. These "experimental autoallergies" include a wide variety of organs and tissues, and are now being employed as model systems for investigation of autoimmune phenomena.

Both humoral antibody and sensitised cells may function in autoimmune disease. Auto-antibodies seem to be involved in reactions with cells which are easily accessible, such as the formed elements of the blood (in haemolytic anaemia, leucopenia, thrombocytopenia), vascular endothelium, vascular basement membrane including the glomerulus (in acute glomerulonephritis) and ascites cells (neoplastic immunity).

Production of lesions in the solid vascularised tissues appears to depend on delayed hypersensitivity reactions with sensitised lymphoid cells (such as in allergic encephalomyelitis, thyroiditis, subacute and chronic glomerulonephritis, orchitis, adrenalitis and many other diseases).

It is quite obvious now that the same autoimmune mechanisms are responsible for the same diseases in human beings and that the extent of such damage is enormous and keeps increasing, with more and more vaccines added to the "recommended" schedule.

Lately, innocent parents and other carers have been accused of causing the symptoms of vaccine damage by allegedly shaking their babies.

Indeed, vaccines such as the pertussis vaccine are actually used to *induce* autoimmune diseases in laboratory animals, the best and most publicised example being the so-called experimental allergic encephalomyelitis (EAE). When, as expected, these unfortunate animals develop EAE from the pertussis vaccine, the causal link is never disputed; yet when babies after vaccination with the same vaccines develop the same symptoms of EAE as the laboratory animals, the causal link to the administered vaccine is always disputed and usually considered "coincidental". Lately, innocent parents and other carers have been accused of causing the symptoms of vaccine damage by allegedly shaking their babies.

Systemic lupus erythematosus is one of the innumerable recognised side-effects of a number of vaccinations. One of the best papers (if not the best) on this is by Ayvazian and Badger (1948), and it has not lost any of its punch and relevance since it was published. They describe three cases of nurses who were literally vaccinated to death. The authors surveyed a group of 750 nurses who trained at a large municipal hospital between 1932 and 1946, and detailed the cases of three nurses who were vaccinated with a multitude of vaccines over a period of time and developed and succumbed to disseminated lupus erythematosus.

Typically, these nurses were given the following tests and vaccines in short succession: the Schick test; three days later, the Dick test; seven days later, typhoid-paratyphoid vaccine; seven days later, another typhoid-paratyphoid vaccine (a double dose); seven days later, the third typhoid-paratyphoid vaccine; and seven days later, the fourth typhoid-paratyphoid vaccine. Every time, the recipient developed local erythema and/or fever and malaise, but it did not deter the doctor from administering yet another series of vaccines, starting only 14 days after the first lot of tests and typhoid-paratyphoid vaccines.

This time, after all these injections, one of the trainee nurses

was given her first injection of scarlet fever streptococcus toxin with "no ill results". One week later, she was given the second injection of streptococcus toxin, after which she developed joint pains and fever. She did not report these reactions to the health office. Nine days later, she returned and received the third injection of a fourfold dose of streptococcus, after which she developed severe arthralgia in the fingers and knees and a sore throat. She was hospitalised for five days and discharged with the diagnosis "Dick-toxin reaction". Only five days later her inoculations were continued, first in lower and then in gradually increasing doses so that the series included a total of 10 instead of the usual seven injections. Epinephrine was administered with each of these injections of streptococcus toxin and toxin-antitoxin.

Two months after the last lot, the trainee nurse was re-admitted to the hospital with swelling and pain of the ankles and toes and tenderness of the joints of both hands, which had been constant since the first Dick test five months earlier. The diagnosis was "rheumatic arthritis". She was given aspirin, but two weeks later the pain came back and she developed chills and fever, sore throat and cough. One month later, the trainee nurse was re-admitted to hospital for two weeks, and during this admission a streptococcus vaccine was started in small doses, but because of her severe reaction "further vaccines were refused". The diagnosis after this admission was "rheumatoid arthritis and infectious mononucleosis". Four months later, the trainee nurse noticed skin eruptions over her nose and both cheeks, and her saliva became foul. The skin and cheeks, upper lips and the bridge of the nose were covered with purplish red, mottled and indurated rash eruptions. Two months later, the eruptions spread over much of the body. A year later, the trainee nurse died, but not before developing severe symptoms of high fever, tachycardia, diarrhoea and showing abnormal blood tests.

It was not enough that this unfortunate trainee nurse died; there were another two cases reported, almost identical to the first case. We shall never know how many of the remaining 747 trainee nurses developed less lethal, but still health-incapacitating, reactions.

If someone said that this type of "medical treatment" had been given to the inmates of the Nazi concentration camps, I would not be surprised. However, this type of "medical treatment" was and *is* being given with impunity to millions of babies, children, teenagers and adults in so-called free and democratic countries as well as in the Third World. Meanwhile, the health authorities refuse to accept that vaccines cause such reactions and even deaths.

VACCINATION: A SAFETY WARNING

The conclusions which follow the study of relevant medical and immunological literature dealing with vaccines and the adjuvants used in vaccines is that the absolute safety of these substances can never be guaranteed. According to Gupta et al. (1993), the toxicity of adjuvants can be ascribed in part to the unintended stimulation of various mechanisms of the immune response. That's why the safety and adjuvancy must be balanced to get the maximum immune stimulation with minimum side effects.

My conclusion is that such balance is impossible to achieve, even if we fully understood the immune system and the full spectrum of deleterious effects of foreign antigens and other toxic substances such as vaccine and drug adjuvants and medications on the immune system of humans, and particularly on the immature immune system of babies and small children. Injecting any foreign substance straight into the bloodstream will only cause anaphylactic (sensitisation) reactions. Nature, over thousands and thousands of years, has developed effective immune responses; yet man, without respect for nature, demonstrably causes more harm than good.

Vaccination procedures are a highly politically motivated non-science, whose practitioners are only interested in injecting multitudes of vaccines without much interest or care as to their effects. Data collection on reactions to vaccines is only paid lip service, and the obvious ineffectiveness of vaccines to prevent diseases is glossed over.

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The fact is that natural infectious diseases have a beneficial effect on the maturation and development of the immune system.

The fact that natural infectious diseases have a beneficial effect on the maturation and development of the immune system is ignored or deliberately suppressed.

Consequently, parents of small children and any potential recipients of vaccines and any orthodox medications should be wary of any member of the medical establishment (which is little more than a highly politicised business system) extolling the non-existent virtues of vaccination. Even though Australian law requires doctors to warn patients about all side-effects of all medications and procedures of a material nature, whether the patient asks or not, doctors as a rule do not uphold this important law. ∞

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Since the mid-1980s when she helped develop the Cotwatch breathing monitor for babies at risk of cot death (sudden infant death syndrome, or SIDS), Dr Scheibner has done extensive research into vaccines and vaccinations. In 1993 she published her book, *Vaccination: The Medical Assault on the Immune System*, and in mid-2000 followed up with *Behavioural Problems in Childhood: Link to Vaccination* (see review, 7/05).

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